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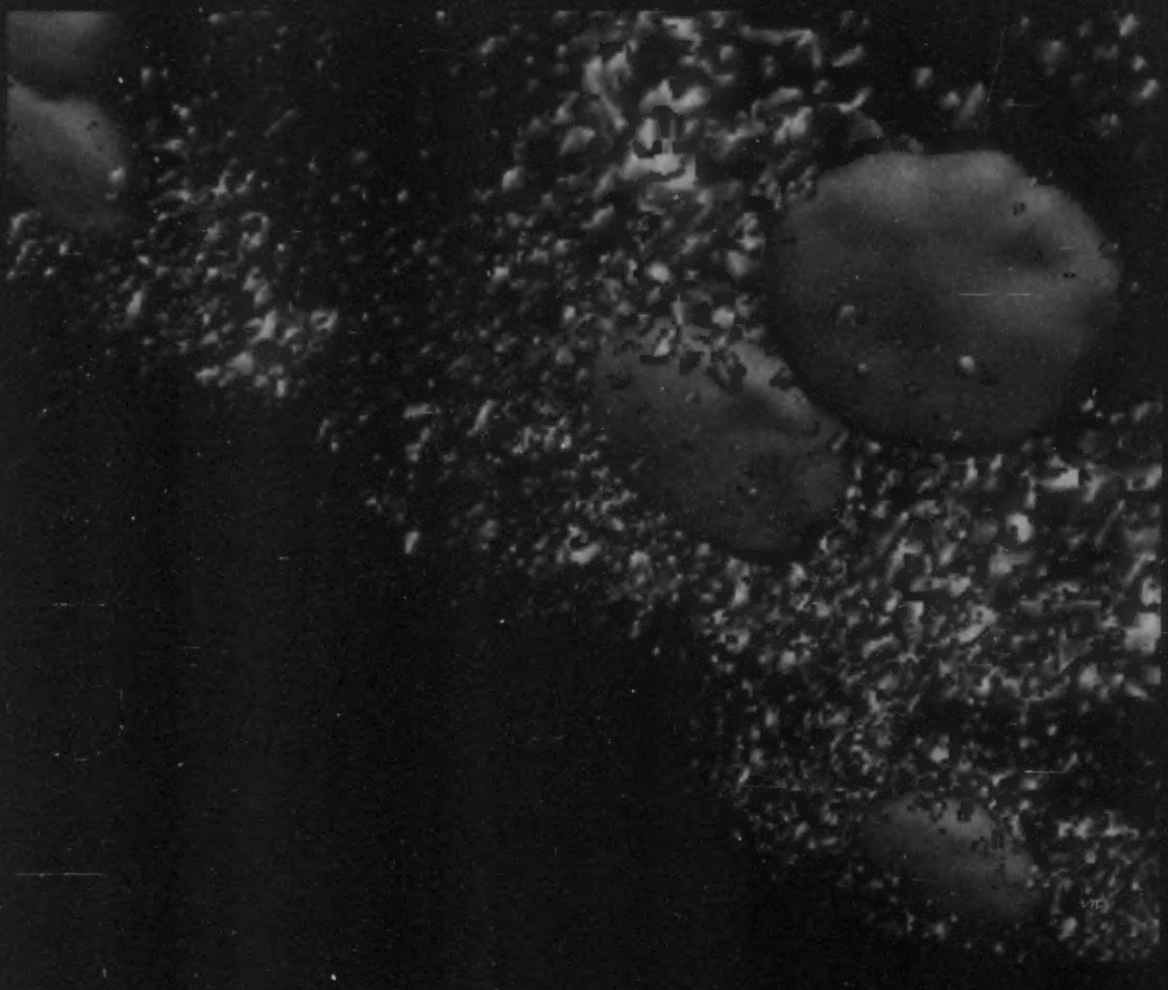

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2003

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The cover picture has kindly been provided by Craig Daily and John Christie McGrath (Glasgow University, Scotland).

The plate shows α_1-adrenoceptors throughout the cell body of hepatocytes with no significant concentration on the cell surface. Binucleated hepatocytes in a mouse liver lobe are visualised by confocal microscopy and a 3D image is reconstructed using IMARIS software. Receptors are shown by the binding of a fluorescent from of an α_1-adrenoceptor antagonist (BODIPY FL-prazosin, blue: excitation 488 nm, detection 515 nm) and nuclei by propidium iodide (pink: excitation 488 nm, detection 550 nm (Daly, C.J. & McGrath, J.C. (2003). *Pharmacol Ther.* 100:101–118; McGrath, J.C. *et al.* (1996). *Trends Pharmacol. Sci.* **17** (11) 393–399.)

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Format	**JPEG** for photographs **GIF** for line drawings or charts
Filenaming	Please save image with .jpg or .gif extension to ensure it can be read by all platforms and graphics packages.

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Format	**JPEG** for photographs **GIF** for line drawings or charts
Filenaming	Please save image with .jpg or .gif extension to ensure it can be read by all platforms and graphics packages.

Authors may be asked to pay the full colour fee for figures that are not submitted in the format described above.

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The purpose of *Special Reports* is to provide rapid publication for new and important results which the Editorial Board considers are likely to be of special pharmacological significance. *Special Reports* will have publication priority over all other material and so authors are asked to consider carefully the status of their work before submission, and to state briefly in the covering letter why they believe their work deserves priority publication.

In order to speed publication there is normally no revision allowed beyond very minor typographical or grammatical corrections. If significant revision is required, the Board may either invite rapid re-submission or, more probably, propose that it be re-written as a Full Paper and be re-submitted for consideration. In order to reduce delays, proofs of *Special Reports* will be sent to authors but **essential corrections must reach the Publisher within 48 hours of receipt**. Authors should ensure that their submitted material conforms exactly to the following requirements.

Special Reports must occupy no more than **four printed pages**. One copy of the manuscript plus a copy on disk should be provided. The manuscript should be prepared in a two-column format as follows; Text (divided into Summary, Introduction, Methods, Results, Discussion, Acknowledgements and References (maximum of 20), all sub-headings in bold) should be justified and in Times New Roman (9 pt). Columns should measure no more than 83 mm (width) and 250 mm (length). The Title (18 pt bold), Authors (12 pt bold) and their Affiliation (9 pt) should spread across both columns. The Summary (9 pt) should measure 140 mm (width) and should not exceed 150 words (plus Keywords and Abbreviations as for Full Papers). Within this format there is no limitation to the number of illustrations (Figures and Tables, with legends) allowed. Authors must supply illustrations which are appropriately sized for direct inclusion in the Journal. These should be either 80 mm width (including axis labelling) to fit single column or 160 mm width (including axis labelling) to fit double column. Authors must ensure that sufficient space is left in the body of the text for these illustrations with their associated legends. All lettering in Figures should be in plain sans serif typeface (Univers, 8 pt). **Please note that the complete manuscript (including text, Figures and Tables prepared as above) must not exceed the four printed page maximum. Manuscripts which do not conform to these requirements may be returned to authors for amendment without review.**

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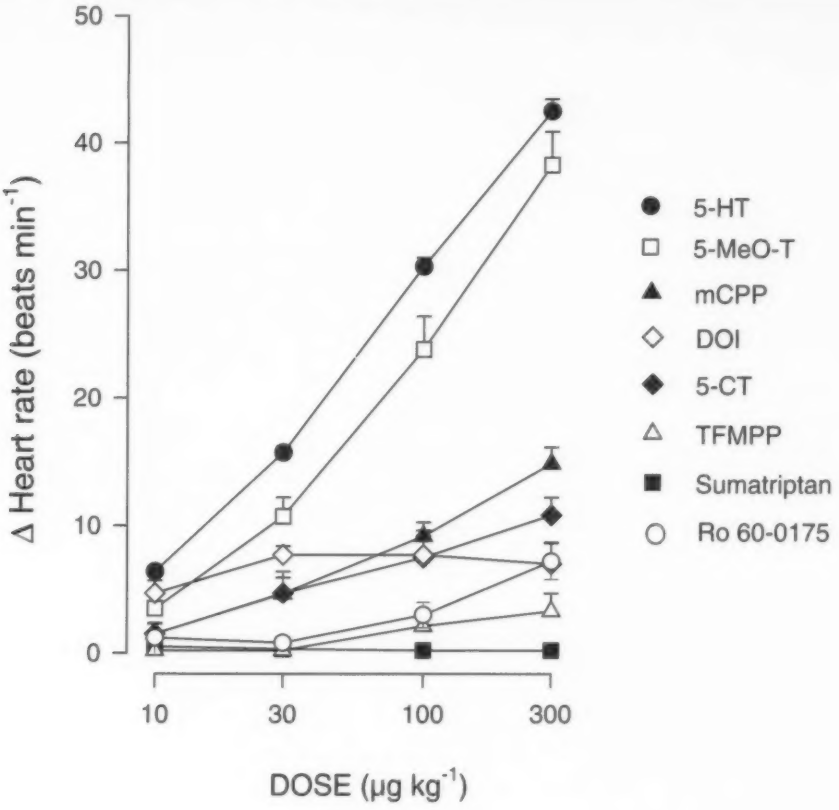
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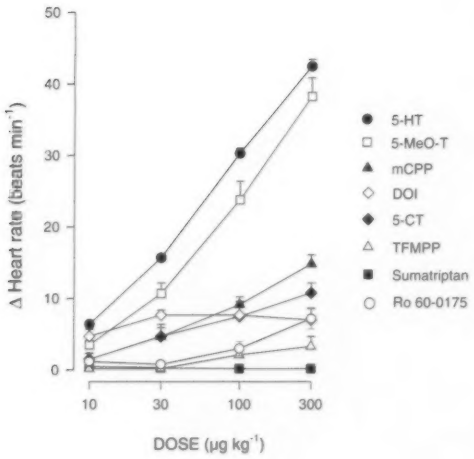
Sumatriptan
Ro 60-0175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5-HT
5-MeO-T
mCPP
DOI
5-CT
TFMPP
Sumatriptan
Ro 60-0175

Figure 1

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A summary of 250 words is required.
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Authors are encouraged to include diagrammatic material or published data records as appropriate.

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ABBREVIATIONS AND SYMBOLS

Physico-chemical quantities

The *British Journal of Pharmacology* uses the SI symbols for units. The following prefixes for multiples of units should be used:

Multiplier	*Prefix*	*Symbol*
10^{-1}	deci	d
10^{-2}	centi	c
10^{-3}	milli	m
10^{-6}	micro	μ
10^{-9}	nano	n
10^{-12}	pico	p
10^{-15}	femto	f
10^{-18}	atto	a
10^{3}	kilo	k
10^{6}	mega	M
10^{9}	giga	G
10^{12}	tera	T

Thus, micron = μm; ångstrom = 0.1 nm. Mixed prefixes are not permissible, thus m μg should be ng. The symbols d (10^{-1}) and c (10^{-2}) should be restricted to those occasions on which there is a strongly felt need for them (e.g. cm).

SYMBOLS

Symbols denoting physical quantities are usually printed as italic capitals (indicated by single underline in typescript). A dash over the symbols indicates a mean value; a dot over the symbol indicates a time derivative. Suffixes may be used to indicate 'where' and 'what'. They are printed as inferiors on that line. Multiple suffixes should be avoided if a simpler symbol adequately defined is unambiguous, but if necessary should be separated by commas e.g. P_{A,CO_2} denotes partial pressure of CO_2 in alveolar air.

CHEMICAL AND BIOLOGICAL ABBREVIATIONS

Authors should also consult *Nomenclature Guidelines for Authors* contained in this issue of the Journal. The abbreviations listed below may be used without definition. Other chemicals, drugs, enzymes and units must be written in full at first mention in the title, summary and again in the text. At first mention they should be followed by the abbreviation in brackets. Subsequently, the abbreviation alone may be used.

Use abbreviations sparingly as extensive use can make the text hard to follow.

Use of solidus

The solidus should be avoided as far as possible and the negative index substituted, e.g. mg kg^{-1} rather than mg/kg; pmol mm^{-2}min^{-1} rather than pmol/mm^2/min.

Physico-chemical quantities

Quantities	*Preferred Unit*	*Symbol*
Amount of substance	mole	mol
Capacitance	Farad	F
Concentration	moles per litre	M or mol l^{-1}
Current	Ampere	A
Electrical conductance	Siemens	S
Electromotive Force	Volt	V
Flow (blood or other liquid)	Litres per second (or min)	l s^{-1} or l min^{-1}
Flow (air or other gas)	Litres per second (or min)	l s^{-1} or l min^{-1}
Force	Newton	N
Frequency	Hertz	HZ
Length	meter	m
Molecular Weight	Dalton	Da
Power	Watt	W
Pressure	Pascal	Pa
Radioactivity	Becquerel	Bq preferred to Ci (3.7×10^{10} Bq)
Resistance	Ohm	Ω
Weight	gram	g

Chemical and biological abbreviations

about	~
acetylcholine	Ach
acetylcholinesterase	AchE
acetyl Coenzyme A	Acetyl CoA
adenosine diphosphate	ADP
adenosine monophosphate	AMP
adenosine triphosphate	ATP
adenosine triphosphatase	ATPase

adenosine 3′5′ cyclic monophosphate	cAMP
adenylyl cyclase	AC
agonist potency	pA_2
alternating current	a.c.
γ-aminobutyric acid	GABA
amino-3-hydroxy-5-methylisooxazole-4-propionic acid	AMPA
analysis of variance	ANOVA
anhydrous	anhyd
angiotensin converting enzyme	ACE
antagonist potency	pD_2
approximate(ly)	approx.
approximately equals	≈
artificial cerebrospinal fluid	aCSF
blood pressure	BP
bovine serum albumin	BSA
cardiovascular system	CVS
catechol-*O*-methyl transferase	COMT
central nervous system	CNS
centrifugal force	*g*
cerebrospinal fluid	CSF
choline acetyltransferase	ChAT
coenzyme A	CoA
complementary deoxyribonucleic acid	cDNA
complementary ribonucleic acid	cRNA
correlation coefficient	*r*
cyclooxygenase	COX
day(s)	d
degree of freedom (statistics)	d.f.
deoxyribonucleic acid	DNA
deoxyribonuclease	DNase
dextro-(absolute configuration)	D-
dextro-(optical rotation)	(+)-
diacyglycerol	DAG
3,4-dihydroxyphenylanine	DOPA
3,4-dihyroxyphenylethylamine	dopamine
direct current	d.c.
disintegrations per minute	dpm
dissociation constant	K_D
dissociation constant, negative logarithm of	pK
edition	edn
editor(s)	ed.
effective concentration 50% of maximum response	EC_{50}
effective dose 50% of maximum response	ED_{50}
electrocardiogram	ECG
electroconvulsive therapy	ECT
electroencephalogram	EEG
electromyogram	EMG
electron spin resonance	ESR
end plate potential	epp
equilibrium constant	*K*
ethylenediaminetetraacetic acid	EDTA
ethylene glycol-bis (β-amino ethyl ether) tetraacetic acid	EGTA
excitatory postsynaptic current	epsc
excitatory postsynaptic potential	epsp
extracellular fluid	ECF
GABA transaminase	GABA-T
gas-liquid chromatography	GLC
glomerular filtration rate	GFR
glutamic acid decarboxylase	GAD
guanosine triphosphate	GTP
guanosine 5′-3′thio-triphosphate	$GTP\gamma S$
guanosine 3′5′ cyclic monophosphate	cGMP
guanylyl cyclase	GC
haemoglobin	Hb
half-life	$t_{1/2}$
high performance liquid chromatography	HPLC
hour or hours	h
human serum albumin	HSA
hydrogen-ion concentration	$[H^+]$
hydrogen-ion activity, negative logarithm of (hydrogen-ion exponent)	pH
6-hydroxydopamine	6-OHDA
N-[2-hydroxyethyl]piperazine-N′-[2-ethanesulphonic acid]	HEPES
5-hydroxytryptamine	5-HT
immunoglobins	IgA;IgD;IgE; IgG;IgM
inhibitor constant	*Ki*
inhibitory concentration (e.g. 50% inhibition of maximum)	IC_{50}
inhibitory postsynaptic current	ipsc
inhibitory postsynaptic potential	ipsp
international unit	iu
intra-arterial	i.a.
intracellular fluid	ICF
intradermal	i.d.
intramuscular	i.m.
intraperitoneal	i.p.
intracerebroventricular	i.c.v.
intrathecal	i.t.
intravenous	i.v.
isotope (atomic mass) e.g. iodine-131	^{131}I
isotopically substituted compounds e.g.	[^{14}C]-ethanol
laevo-(absolute configuration)	L
laevo-(optical rotation)	(−)-
lethal dose (in 50% of population)	LD_{50}
lipoxygenase	LOX
magnetic resonance imaging	MRI
maximum	max.
mean arterial pressure	MAP
mean value of (statistics)	$\bar{x}$
messenger ribonucleic acid	mRNA
Michaelis constant	K_M
miniature end plate potential	mepp
minimum	min.
minute or minutes	min
monoamine oxidase	MAO
nitric oxide	NO
nitric oxide synthase	NOS
N-methyl-D-aspartate	NMDA
non-specific binding	NSB
nuclear magnetic resonance	NMR
number	no. or No.
number of observations (statistics)	*n*
optical density	OD
oral	p.o
page/pages	p/pp
parts per million	p.p.m.
per cent	%
phenylethanolamine N-methyltransferase	PNMT
phosphatidyl inositol	PI
phosphodiesterase	PDE
phospholipase (A_2,C,D)	PL(A_2,C, D)
polymerase chain reaction	PCR
probability (significance level in a statistical test)	*P*
protein kinase (A,B,C,G)	PK (A,B,C,G)
radiommunoassay	RIA
renal blood flow	RBF
ribosomal RNA	rRNA
revolutions per minute	rpm
ribonucleic acid	RNA
second or seconds	S
somatostatin	SST
standard deviation (of observed sample)	s.d.
standard error (of estimate mean value)	s.e.mean
subcutaneous	s.c.
sum (statistical):	
of hypothetical population	Σ
of observed sample	S or Σ

thin layer chromatography	TLC	ultraviolet	u.v.
time, clock 24 h clock used e.g 18 h 30 min	t	unit	u
time constant	τ	volume by volume	$v\,v^{-1}$
2-amino-2-hydroxymethyl-propan-1,3,- diol	Tris		
transfer ribonucleic acid	tRNA	wavelength	λ
tyrosine hydroxylase	TH	weight	wt.
tyrosine kinase	TK	weight by volume	$w\,v^{-1}$

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NOMENCLATURE GUIDELINES FOR AUTHORS

With effect from February 2004

The Nomenclature Working Party (NWP) of the Editorial Board of the British Journal of Pharmacology has consulted many acknowledged experts in an effort to clarify and standardise receptor and other nomenclature systems for use by Editors until a complete set of recommendations from the International Union of Pharmacology Committee on Receptor Nomenclature and Drug Classification (NC-IUPHAR) is published.

The NWP is unanimous in its view that with rare exceptions, the Journal should use spellings, names and abbreviations that have been chosen by international bodies convened for the purpose.

For receptor nomenclature, with few exceptions, the Journal generally follows the guidelines laid down in two sources, the IUPHAR Receptor Compendia and the reports of NC-IUPHAR Sub Committees published in *Pharmacological Reviews*. In the following Sections, these sources will be referred to as *IUPHAR* and *Pharmacol. Rev.*

1 *Definition of receptors and subtypes*

Receptors and their subtypes are defined in terms of structural information where available, affinity and selectivity of antagonists and thereafter by agonist selectivity.

2 *Format of receptor names*

(a) Editors will permit with reluctance new nomenclature systems in papers accepted for publication if, and only if, there are compelling reasons to introduce a new terminology (or modify an accepted one). The criteria upon which the new receptor type or subtype are defined must be given, together with adequate explanations of the relationship between the previous nomenclature (fully referenced) and the proposed one.

N.B. The new nomenclature should not appear in the Title, Short Title or Keywords, unless qualified by the adjective putative (e.g.... mediated by the putative imidazoline I_2 receptor).

(b) Only well-established and universally accepted subtype names will be acceptable without any reference to the originator of these terms. In cases of controversy concerning further subdivision of the subtype, full referencing must be given.

(c) When receptors are expressed from DNA or RNA that has been introduced into cells and these receptors display a dissimilar pharmacological profile to the native receptors, or have not been characterised pharmacologically, they should be denoted by use of lower case, *e.g.* 5-ht_5 for expressed receptors and 5-HT_7 for native receptors. The stoichiometry of the expressed receptor should be indicated, where appropriate, e.g. for an adult muscle nicotinic acetylcholine receptor, it might be $(\alpha_1)2\beta_1\gamma\delta$.

(d) Receptor subtypes should be designated by means of a subscript numeral or capital letter. Some double subscripts (i.e. numeral plus letter) are acceptable.

(e) Greek letters and Roman numerals should be avoided in any new nomenclature. The name should not include the letter 'R' or 'r' as an abbreviation for receptor.

(f) Mammalian systems are the basis of receptor classifications with primacy given to humans. Therefore non-mammalian species should be clearly indicated, e.g. *Torpedo* nicotinic acetylcholine receptor, turkey β adrenoceptor, locust GABA receptor.

3 *Types of receptor*

The NWP accepts that there are additional receptors to those described below which can be considered to be well characterised. In many cases, however, their existence has been confirmed only in cloning studies and it is as yet unclear how they relate to similar subdivisions proposed on the grounds of differences in agonist and antagonist potencies in various tissues. Italics should be used if the receptor has not been cloned. Lower case should be used to describe cloned receptors for which endogenous expression has not been described. It is incorrect to refer to any receptor, using the suffix 'ergic' (*e.g.*, cholinergic, adrenergic). See Section 5 for the correct use of such terms.

(1) Acetylcholine receptors The two principal subfamilies are muscarinic and nicotinic acetylcholine receptors.

Muscarinic acetylcholine receptors The principal subtypes are M_1, M_2, M_3, M_4 and M_5. (See Caulfield, M.P. and Birdsall, N.M.J. (1998) *Pharmacol. Rev.*, **50**, 279-290.

Nicotinic acetylcholine receptors Receptors should be described as nicotinic (not n), and where it is known the stoichiometry should be given at first mention. NC-IUPHAR recommend that nicotinic receptor subtypes should be classified according to the predominant α subunit (α_1–α_{10}) contained within the receptor complex. (See Lukas, R.J. *et al.*, (1999) *Pharmacol. Rev.*, **51**, 379–401).

(2) Adenosine receptors Principal subtypes are A_1, A_{2A}, A_{2B} and A_3. (See Fredholm, B.B. *et al.*, (2001) *Pharmacol. Rev.*, **53**, 527–522).

(3) Adrenoceptors Principal subtypes are α_{1A}, α_{2A}, β_1, β_2, and β_3 adrenoceptors. Additional subtypes must be fully referenced. (See Bylund, D.B., *et al.* (1994) *Pharmacol. Rev.*, **46**, 121–136 and Hieble, J.P., *et al.* (1995) *Pharmacol. Rev.*, **47**, 267–270).

For further subdivision, please refer to Bylund, D.B., *et al.* (2000) *IUPHAR compendium of receptor characterisation and classification, 2nd Edition*, pp. 88-103 IUPHAR media, London.

(4) Angiotensin receptors The principal subtypes are AT_1 and AT_2. (See de Gasparo M., *et al.* (2000) *Pharmacol. Rev.*, **52**, 415–472).

(5) Atrial natriuretic peptide receptors The principal subtypes are ANP_A and ANP_B.

(6) Bombesin receptors The principal subtypes are BB_1, BB_2 and BB_3.

(7) Bradykinin receptors The principal subtypes are B_1 and B_2.

(8) Calcitonin gene-related peptide (CGRP) receptors The principal subtypes are calcitonin (CT), amylin (AMY), CGRP, and adrenomedullin (AM). (See Poyner, D.R. *et al.* (2002) *Pharmacol. Rev.*, **54**, 161–202).

(9) Cannabinoid receptors The principal subtypes are CB_1 and CB_2. (See Howlett, A.C., *et al.* (2002) *Pharmacol. Rev.*, **54**, 161–202).

(10) Chemokine receptors The principal subgroups are XCR, CCR, CXCR, CX_3CR. Subtypes within these subgroups must be fully referenced (See Murphy, P.M., *et al.* (2000) *Pharmacol. Rev.*, **51**, 145–168 and Murphy, P.M. (2002). *Pharmacol. Rev.*, **54**, 227–229).

(11) Cholecystokinin (CCK) receptors The principal subtypes are CCK1 and CCK2. (See Noble, F., *et al.* (1999) *Pharmacol. Rev.*, **51**, 745–781).

(12) Corticotrophin receptors The principal subtypes are CRF_1 and CRF_2. (See Hauger, R.L., *et al.* (2003) *Pharmacol. Rev.*, **55**, 21–26).

(13) Dopamine receptors The principal subtypes are Dl, D2, D3, D4 and D5. (See Schwartz J-C., *et al.* (2000) *IUPHAR compendium of receptor characterisation and classification, 2nd Edition*, pp. 171-181, IUPHAR media, London).

(14) Endothelin receptors The principal subtypes are ET_A and ET_B. (See Davenport, A.P. (2002) *Pharmacol. Rev.*, **54**, 219–226).

(15) Galanin receptors The principal subtypes are GAL_1, GAL_2 and GAL_3. (See Branchek, T. *et al.* (2000) *Trends Pharmacol. Sci.*, **21**, 109–116).

(16) γ-Aminobutyric acid (GABA) receptors The principal subtypes are $GABA_A$ and $GABA_B$. Modulatory sites on either receptor type should be fully referenced. (See Barnard, E.A. *et al.* (1998) *Pharmacol. Rev.*, **50**, 291–313; Bowery, N.G. *et al.* (2002) *Pharmacol. Rev.*, **54**, 247–264).

(17) Glucagon-like peptide receptors The principal subtypes are GLP1 and GLP2. (See Mayo, K.E., *et al.* (2003) *Pharmacol. Rev.*, **55**, 167–194).

(18) Glutamate receptors Three ionotropic subtypes are recognised and named: (1) NMDA receptors; (2) AMPA receptors, and (3) kainate receptors. The metabotropic glutamate (mGlu) receptor are subdivided into mGlu 1,2,3,4,5,6,7,8. Modulatory sites must be fully referenced. (See Schoepp, D.D., *et al.* (2000) *IUPHAR compendium of receptor characterisation and classification, 2nd Edition*, pp. 195–206, IUPHAR media, London).

(19) Glycine receptor When it is known the stoichiometry should be given at first mention. A distinction should be drawn between the immature receptor composed of α2 subunits expressed by embryonic neurons and the mature receptor containing predominantly α1 and β subunits expressed by adult neurons.

(20) Histamine receptors The principal subtypes are H_1, H_2, H_3 and H_4 (See Hill, S.J. (2000) *IUPHAR compendium of receptor characterisation and classification* pp. 227–232, IUPHAR media, London).

(21) 5-Hydroxytryptamine (5-HT) receptors The principal subtypes are $5\text{-}HT_1$, $5\text{-}HT_2$, $5\text{-}HT_3$, $5\text{-}HT_4$, $5\text{-}ht_5$, $5\text{-}HT_6$ and $5\text{-}HT_7$. Further subdivisions have been defined (See Martin, G.R., *et al.* (2000) *IUPHAR compendium receptor characterisation and classification*, 2nd Edition, pp. 233–251, IUPHAR media, London).

(22) Inositol(1,4,5)-trisphosphate (Ins(1,4,5)P_3) receptor The principal subtypes are $IP_3$1, $IP_3$2 and $IP_3$3.

(23) Leukotriene (LT) receptors The principal subtypes are BLT_1, BLT_2, $CysLT_1$, and $CysLT_2$. (See Brink, C., *et al.* (2003) *Pharmacol. Rev.*, **55**, 195–227.)

(24) Lysophospholipid (LPL) receptors The principal subtypes are LPA_1, LPA_2, LPA_3 and SIP_1, SIP_2, SIP_3, SIP_4, SIP_5 (See Chun, J. *et al.* (2002) *Pharmacol. Rev.*, **54**, 265–269).

(25) Melanocortin receptors The principal subtypes are MC_1, MC_2, MC_3, MC_4 and MC_5.

(26) Melatonin receptors The principal subtypes are MT_1, MT_2 and MT_3.

(27) Neuropeptide Y receptors The principal subtypes are Yl, Y2, Y4, Y5 and y6. (See Michel, M.C. *et al.* (1998) *Pharmacol. Rev.*, **50**, 143-150).

(28) Neurotensin receptors The principal subtypes are NT_1 & NT_2.

(29) *Neurotrophin receptors* The principal subtypes are trkA, trkB, trkC and p75.

(30) *Opioid and opioid-like receptors* The principal subtypes are δ (DOP), κ (KOP), μ (MOP) and ORL1 (NOP). Other proposed subtypes should be fully referenced.

(31) *Peroxisome proliferator activated receptors* The principal subtypes are PPAR-α, PPAR-β and PPAR-γ.

(32) *Prostanoid receptors* The principal types are DP, EP, FP, IP and TP. When first mentioned, the style prostanoid (XP) receptor should be used, thereafter, XP receptor (where X denotes the type). Proposed subtypes should be referred to as XP_n (e.g. EP_1, EP_2) and referenced (See Coleman, R.A. *et al.* (1994) *Pharmacol. Rev.*, **45**, 205–229; Coleman, R.A. *et al.* (2000) *IUPHAR compendium of receptor characterisation and classification, 2nd Edition*, pp. 337–353, IUPHAR media, London).

(33) *Protease activated receptors* The principal subtypes are PAR1, PAR2, PAR3 and PAR4. (See Hollenberg, M.D. and Compton, S.J. (2002) *Pharmacol. Rev.*, **54**, 203–217).

(34) *P2X receptors* The principal subtypes are $P2X_{1,2,3,4,5,6,7}$. (See Khakh, B.S. *et al.* (2001) *Pharmacol. Rev.*, **53**, 107–118).

(35) *P2Y receptors* The principal subtypes are $P2Y_{1,2,4,5,6,11,12,13}$. (See King, B.F. *et al.* (2000) *IUPHAR compendium of receptor characterisation and classification, 2nd Edition*, pp. 306–320, IUPHAR media, London).

(36) *Ryanodine receptors* The principal subtypes are RY_1, RY_2 and RY_3.

(37) *Somatostatin (SST) receptors* The principal types are $SRIF_1$ and $SRIF_2$. Proposed subtypes should be fully referenced (See Hoyer, D. *et al.* (2000) *IUPHAR compendium of receptor characterisation and classification, 2nd Edition*, pp. 354–364, IUPHAR media, London).

(38) *Tachykinin (Neurokinin-NK) receptors* The principal subtypes are NK_1, NK_2 and NK_3.

(39) *Vanilloid receptor* The principal subtype is TRPV1 (formerly VR1). Other proposed subtypes should be fully referenced.

(40) *Vasoactive intestinal peptide and pituitary adenylate cyclase activating peptide receptors* The principal subtypes are $VPAC_1$,$VPAC_2$ and PAC_1. (See Harmar, A.J. *et al.* (1998) *Pharmacol. Rev.*, **50**, 265-270).

(41) *Vasopressin and oxytocin receptors* The principal subtypes are V_{1A}, V_{1B}, V_2 and OT. Further subtypes should be fully referenced.

4 *Naming of ion channels*

Ion channels are typically described by an abbreviation of the ion permeating the channel (e.g. K^+ channel, Na^+ channel, Cl^- channel, Ca^{2+} channel etc.) Ionic currents are referred to by either the full description of the current (e.g. Ca^{2+} -activated K^+ current) or as an abbreviation using the prefix *I* followed by the atomic species carrying the current as a subscript (e.g. I_{Na}, I_{Ca},) Where it is important to specify the activator of the current, this may be added to the atomic species subscript in parenthesis (*e.g.* $I_{K(Ca)}$) for a calcium-activated potassium current or $I_{K(V)}$ for a voltage-activated potassium current).

When a pharmacological agent is used to describe a current, it must be fully defined in the text.

When, for example, a system has two currents carried by the same ionic species and activated by similar means but with (for example) different kinetics, the distinguishing factor may be added to the subscript or as a hyphen after the abbreviation (*e.g.* '$I_{K(Ca,slow)}$' or '$I_{K(Ca)}$-slow', which would refer to a kinetically slow calcium-activated potassium current).

Examples of some commonly used abbreviations are shown below:

Sodium current: $I_{Na(V)}$
Potassium currents: $I_{K(V)}$, $I_{K(Ca)}$, $I_{K(A)}$, $I_{K(IR)}$
Chloride currents: $I_{Cl(Ca)}$, $I_{Cl(cAMP)}$, $I_{Cl(swell)}$
Calcium currents: $I_{Ca(L)}$, $I_{Ca(T)}$, $I_{Ca(N)}$

5 *Naming of nerve fibres*

Many nerve fibres are now known to release more than one transmitter, and future work may show that this is, in fact, the general rule. In that case, the concept of the same transmitter being released either at different developmental stages or under various experimental conditions would no longer hold, and single adjectives that imply this (e.g. cholinergic, noradrenergic) would become inappropriate when applied to nerve fibres, as distinct from transmitter functions. For the present, those nerve fibres that are known to function by releasing more than one identified transmitter may be described accordingly; for example, cholinergic-peptidergic (in alphabetical order, the order implying no priority of function). N.B. The suffix 'ergic' should continue to be applied only to nerve fibres and to the transmission event, in accordance with Dale's intentions. For example 'cholinergic' indicates that the nerve fibre, or the transmission, functions through the release of a choline-like substance. The suffix should not be used loosely to mean 'pertaining to'. Hence the expression 'cholinergic receptor' (rather than acetylcholine receptor) is incorrect. Transmission events involving nitric oxide may be referred to as *nitrergic*.

Nerve fibres that release noradrenaline are to be described as noradrenergic. The term adrenergic should be reserved for nerve fibres known to release adrenaline. Where the identity of the catecholamine is uncertain catecholaminergic should be used. The adjective to be applied to nerve fibres that release dopamine as a transmitter is dopaminergic (not DAergic).

NANC is an acceptable abbreviation of non-adrenergic, non-cholinergic for peripheral efferent nerve fibres when the identity of the transmitter(s) is unknown.

Glutamatergic, not glutaminergic, should be used to describe nerve fibres releasing glutamate. In referring to peptide-releasing nerve fibres (e.g. those that may release substance P or vasoactive intestinal peptide) the nomenclature to be used is peptidergic (X), e.g. peptidergic (SP).

The terms 5-hydroxytryptamine (5-HT) and 5-hydroxytryptaminergic (i.e. nerves releasing 5-hydroxytryptamine) are preferred to those of serotonin and serotoninergic.

The term 5-HTergic is not acceptable.

The term purinergic defines transmission mediated by ATP.

6 *Terms used to describe agonist and antagonist action*

The following terms can be used without full definition Where appropriate, other terms may be used but must be accompanied by a full definition.

Terms used to describe affinity and potency

(a) *EC_{50}* The concentration of an agonist that produces 50% of the maximal response for that agonist *in vitro*. The agonist may be stimulatory or inhibitory. When EC_{50} values are determined in the presence of other agonists or antagonists the concentration of the latter should be stated. Related terms, e.g. EC_{25}, are acceptable if accompanied by a full definition.

(b) *IC_{50}* This term may be used in the following ways.

(i) The concentration of antagonist that reduces the response to a sub-maximal concentration of agonist by 50%; the concentration of agonist should be stated.

(ii) The concentration of competing agonist or antagonist that inhibits the binding of a radioligand by 50%; the concentration of radioligand should be stated.

(c) *ED_{50}* This term may be used in the following ways.

(i) The dose of an agonist or antagonist that produces 50% of the maximal possible effect of that agonist or antagonist *in vivo*.

(ii) The dose of drug that produces the effect under investigation in 50% of the population.

(d) *K* The dissociation equilibrium constant (mol l^{-1}), for ligand receptor interactions. The reciprocal is called the affinity constant or association equilibrium constant. When necessary for clarity, subscripts (letters or numerals, or a combination of both) may be added but these must be clearly explained when first used.

(e) *n_H* The Hill coefficient.

(f) *pA_2* The negative logarithm to base 10 of the concentration of an antagonist that makes it necessary to double the concentration of agonist needed to elicit a given submaximal response. Note that the definition is empirical and does not pre-suppose the mechanism of antagonism. The pA_2 value can be determined from a Schild plot with unconstrained slopes, but only provides an estimate of the pK_B if the antagonism has been shown to meet all of the criteria of competition.

(g) *pD_2* The negative logarithm to base 10 of the EC_{50}

(h) *pIC_{50}* The negative logarithm to base 10 of the IC_{50}.

(i) *pK* The negative logarithm to base 10 of K (with or without subscripts as appropriate: see under K above).

Terms used to describe the mode of antagonism

(a) *Competitive antagonism* In competitive antagonism the binding of agonist and antagonist is mutually exclusive. This may be because the agonist and antagonist compete for the same binding site or combine with adjacent sites that overlap. A third possibility is that different sites are involved but they influence the receptor macromolecule in such a way that agonist and antagonist molecules cannot be bound at the same time.

(b) *Irreversible competitive antagonism* Used to describe antagonists that bind irreversibly.

(c) *Non-competitive antagonism* Agonist and antagonist can be bound simultaneously; antagonist binding reduces or prevents the action of the agonist.

(d) *Un-competitive antagonism* Antagonist binding is dependent upon prior agonist activation (*e.g.* open channel blockade).

(e) *Irreversible non-competitive antagonism* Used to describe non-competitive antagonists that bind irreversibly.

For a more detailed account of the terms used to describe agonist and antagonist action see Jenkinson, D.H., *et al.* (1995) *Pharmacol. Rev.*, **47**, 225–266.

7 *Guidance on statistical analysis of data*

Given that populations of data with a Gaussian (normal) distribution on a logarithmic scale (e.g. pD_2, pEC_{50}, etc.) will show a skewed distribution once converted to a natural scale, parametric analysis is only appropriate on the logarithmic scale for such data. Thus, the expression of EC_{50} and ED_{50} values (derived from log concentration-or log dose-response curves) as means$\pm$s.e.mean is inappropriate. However, pD_2 or pIC_{50} values may be reported as means$\pm$s.e.mean.

8 *Enzymes*

The International Union of Biochemistry and Molecular Biology Enzyme Commission (EC) number and full name (Enzyme Nomenclature 1992, Academic Press, San Diego and London) must be quoted when first mentioned in text.

Subsequently the accepted trivial name is used. Trivial names may be used in the title. The following abbreviations may be used; cyclo-oxygenase (COX-1 and COX-2), guanylyl cyclase (GC), monoamine oxidase (MAO-A and MAO-B), nitric oxide synthase (eNOS, nNOS and iNOS), phosphodiesterase (PDE1 to PDE11), catechol-O-methyl transferase (COMT), adenylyl cyclase (AC), acetylcholinesterase (AchE), acetyl coenzyme A (AcetylCoA), adenosine triphosphatase (ATPase), angiotensin converting enzyme (ACE), choline acetyltransferase (ChAT), coenzyme A (CoA), deoxyribonuclease (DNAse), GABA transaminase (GABA-T), glutamic acid decarboxylase (GAD), lipoxygenase (LOX), phenylethanolamine N-methyltransferase (PNMT), phospholipase (PLA_2, PLC, PLD), protein kinase (PKA, PKB, PKC, PKG), tyrosine hydroxylase (TH), tyrosine kinase (TK), ribonuclease (RNase)

9 *Transporters*

Transporters should be defined in full at first mention.

10 *Other nomenclature requirements*

(a) *Racemates* Authors must state unambiguously in the Methods section of papers which isomers were used, e.g. (+)- or (−)-propranolol, and must bring to the attention of the reader the composite character of drugs that are mixtures of stereoisomers. Furthermore, the implications of the composite nature of such drugs studied for the interpretation of the data measured and the conclusions drawn must be made explicit. Capital R and S refer to the absolute configurations of chiral centres and should be used where necessary.

(b) *Purines* This term should not be used as a synonym for purine nucleotides or nucleosides.

(c) *Eicosanoids* The system of nomenclature to be used for eicosanoids is that published in *Methods in Enzymology* (1990) **187**, 1–9. In manuscripts, the first use of the full chemical name of any eicosanoid should indicate double bond geometry when this is known.

(d) *Cell lines* Cell type, range of passage number, species and source should be defined.

(e) *Molecular biology* Abbreviations pertaining to molecular biological techniques need to be defined or presented in such a way that they can be recognised by the non-specialist.

(f) *Tension* Tension is force and should be calibrated in Newtons. ($1\ \text{Newton} = 1\ \text{kg ms}^{-1}$) or in kg weight, g weight, or mg weight etc. It should not be calibrated in units of mass (e.g. kg). (See Miller D.J. (1988) *Trends Pharmacol. Sci.*, **9**, 124–125).

(g) *Ions* When referring to ions, the charge should be indicated, *e.g.* Na^+, Ca^{2+}, $3Na^+/Ca^{2+}$ exchange, etc.

(h) *Inhibitors of nitric oxide synthase (NOS)* Commonly used inhibitors of NOS and their appropriate abbreviations include L-N^G-mono-methylarginine (L-NMMA), L-N^G-nitroarginine (L-NOARG), and L-N^G-nitroarginine methyl ester (L-NAME).

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